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Retinal vascular changes as a diagnostic aid : with special reference to certain hypertensive states

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RETINAL VASCULAR CHANGES AS A DIAGNOSTIC AID WITH SPECIAL REFERENCE TO CERTAIN HYPERTENSIVE STATES

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complex, which are at our disposal. We do not ques-

tion their value. For the most part we are well aware of their significance, yet it is seldom, if ever, that we consider the hardships, the reverses, and the romance which have been a part of their development. For any practitioner to be fully appreciative of any piece of mechanical equipment at his disposal it is necessary that he learns of its' development, that he endeavors to understand that it was once merely an idea in man's mind, and how after its' invention it has been improved, been made more practical, and subsequently has been made available to a greater number through refinements in its' manufacture.

It is almost inconcievable to think of any doctor not availing himself of a stethoscope and utilizing it many times a day. Similarly the ability to interpret x-ray findings is essential, even to the general practitioner. Certainly to say that the rectal speculum is of no assistance in studying pathology affecting that portion of the colon is folly. Fortunately the vast majority of men are willing to admit this and employ these instruments in giving their patients greater assurance that the true nature of their a fliction will be learned before attempts are

made to eradicate it.

This brings me to the principal aim in this thesis; to endeavor to show that the ophthalmoscope is an instrument which no truly conscientious medical practitioner can consider unessential. Very few doctors, save the ophthalmologists, deem the ophthalmoscope of sufficient value in their daily work to warrant their learning to use it. The most plausible reason for this is that they feel that not enough can be learned in the examination of the fundus oculi to make the proceedure worth while. I will endeavor to show, on the contrary, using a considerable amount of literature concerning the subject, to prove that changes in the vasculature of the retina are of great import in visualizing pathologic states far remote from the eye.

Due to the enormous amount of literature written on this subject it has been necessary to limit the scope of this presentation to certain hypertensive states as they manifest themselves in the eyegrounds. Therefore, it should be bourne in mind that this is in no sense intended to be a complete review of all literature concerning circulatory disturbances in the retina and their significance in indicating pathology

HISTORY

The ancients observed the luminosity of the eyes of certain animals for there is mention of it by Aristotle, and Pliny says "the eyes of nocturnal animals such as cats, are brilliant in the darkness." Similar observations were later made in the dog, horse, sheep, weazel, hyena, and birds of prey(27).

In 1704, Mery having accidently held a cat under water, distinctly observed the color of the bottom of the eye, and the blood vessels ramifying thereon(41). La Hire in 1709 explained the phenomenon as follows: "When a normal eye is in the air the rays of light issuing from a point in the fundus are so refracted that they leave the eye in parallel lines. For this reason we should be able to see the point in the fundus clearly, for parallel or almost parallel rays always produce a distinct perception in our eye; nevertheless, we do not see the object. On the other hand, when the eye is under water the rays leaving the eyeball diverge and in passing from the water into the air they are made to diverge still more. The result is that , wherever we place our eye these divergent rays give us a clear picture of the point in the fundus from which they emerge".

He does not attempt to explain the problem why the parallel rays emerging from an eye exposed to the air cannot be seen.

The first mention of the observation in the human eye was made in 1796 by Fermin who saw that the pupils of an Ethiopian albino were luminous.

The bright yellow appearance of the pupils in certain forms of disease, first mentioned by Scarpa in 1816, was classically described by Beer in 1817 under the title "Amaurotic Cat's Eyes".

We find no mention of luminosity in other than albinotic or diseased human eyes until 1837 when Behr observed it in a case of total irideremia and it was not until the forties before the observation was made on normal eyes.

It is interesting to learn the theories to explain these observations. First it was regarded as a phenomenon of phosphoresence, by some as the light absorbed during the day and given off at night and later by others as the result of and internal activity similar to that of the firefly. It was described as varying with the seasons, with the age of the individual and with his nervous state. Electricity was also called upon to assist in explaining the luminosity of the

eye. It was the "naked electricity emitted by the retina, for nowhere in the animal organism is the brain substance exposed to the naked eye as clearly as in the open interior of the eyeball"(Pallas, 1811).

But Prevost in 1818 pointed out the true course of the reflection of the light which entered the eye, and Gruithuisen about the same time came to a similar conclusion. In 1821 Rudolphi added the observation that success of the experiment depended upon having the light thrown in, in a definite direction and that the eyes of the decapitated head of a cat were as easily made luminous as in the living.

Esser in 1826 showed that such eyes show even brighter than the living because of the larger size of the pupil. In 1836 Hasenstein showed that he could make the pupil luminous by compressing the eyeball in its' anteroposterior diameter, and in 1845 Brueke gave the correct explanation of the red color of the luminous pupil in that the light was reflected by the choroidal blood vessels(27).

Dr. Kussmaul, in our essay on colored appearances at the bottom of the human eye, published in Heidelberg in 1845, endeavored to explain how it is that the interior of the eye ordinarily appears dark and that the bottom is not seen. In illustration of the influence

of the refractive media on the visibility of the bottom of the eye, he adduced the following experiment: Take an eye-a sheeps eye will do-and remove the cornea. On looking toward the bottom it will be seen dark, because the retina lies nearly in the focus of the lens; but as soon as this body is removed, the retina and its' blood vessels become visible. That the position of the retina within or without the focus is the cause of its' not being visible, he proved by removing a part of the vitreous humor. By this the lens approached the retina, which thus came to lie within the focus, and was seen as if through a convex glass magnified. Herein is afforded an explanation of the cases of far-sightedness in old persons in which the entrance of the optic nerve is visible at the bottom of the eye. By atrophy of the eyeball in like manner the retina comes to be distinctly seen(41).

Cumming in 1846, and Brueke in 1847 made observations similar to those of Kussmaul by transmitting light into the eye in a concentrated beam and studying the directions of the beam as it was reflected from the eye(68).

In 1847 Dr. Von Erlach, who wore spectacles noticed that the eyes of other persons appeared to

him to shine when the person observed saw the images of the lamp flame reflected in Dr. Von Erlack's spectacles. In this accidental observation it will be seen lies the principle of the ophthalmoscope(41).

In 1844 Babbage contrived a crude instrument for looking into the interior of the eye. It consisted of a bit of plain mirror, with the silvering scraped off at two or three small spots in the middle, fixed within a tube at such an angle that the rays of light falling on it through an opening in the side of the tube, were reflected into the eye to be observed, and to which the one end of the tube was directed. The observer looked through the clear spots of the mirror from the other end(26).

La Hires profound statement(1909) already referred to, was too advanced, others receded from it and it required almost 150 years before the problem was solved.

In 1851, a little pamphlet was published by Helmholtz, then a young professor of anatomy and physiology in Koenigsberg, under the title of "Beshreibung Eines Augen Spiegels zur Untersuchung der Netzhaut im Lebenden Auge". In this he demonstrated the fundamental fact that the rays pass out of the

eye in the same lines in which they have entered. He explained many of the observations of his predecessors which heretofore had not been understood, but what was more important, he added the practical to the theoretical and described an instrument with which the details of the retina could be examined. He described the ophthalmoscopic appearance of the retina, calculated the enlargement under which it is seen, pointed out the value of the instrument as a measure of refraction and the accomodative changes of the eye. His short monograph was thorough and complete and gave into our hands a means of examination of which no one had yet dreamed.

In his modest way Helmholz thus prophesies its' usefullness, "I do not doubt, judging from what can be seen of the state of the healthy retina, that it will be possible to discern all its' diseased conditions, so far as these, if seated in other transparent parts, such as the cornea, would admit of diagnosis by the sense of light. Distention or varicosity of the retinal vessels will be easily perceptible".

The invention of this instrument ushered in a new era in ophthalmology, the most prolific era in the history of this science. The influence it has

wielded upon other branches of medicine is far-reaching. It will not be out of place to tell the story of the invention of the instrument in Helmholz's own words, "I was endeavoring to explain to my pupils the emission of reflected light from the eye, a discovery made by Brueke, who would have invented the ophthalmoscope had he only asked himself how an optical image is formed by the light returning from the eye. In his research it was not necessary to ask it, but had he asked it, he was just the man to answer it as quickly as I did and to invent the instrument. I turned the problem over and over to ascertain the simplest way in which I could demonstrate the phenomenon to my students. It was also a reminiscence of my days of medical study that ophthalmologists had great trouble in dealing with certain cases of eye disease then known as black cataract. The first model was constructed of pasteboard, eye lenses, and cover glasses used in the microscopic work. It was at first so difficult to use that I doubt if I should have persevered, unless I had felt that it must succeed: but in eight days I had the great joy of being the first who saw before him the living human retina(27).

In 1852 Reute developed the idea of indirect

ophthalmoscopy(68).

In 1856 Coote began using the ophthalmoscope in St. Bartholomew's Hospital in London and praised its' value in diagnostics. He was criticized by many who said that it would never be practical but he defended its' use vigorously reminding his colleagues that only a few years prior they had scoffed at the first attempts to devise a stethoscope.

Simultaneously Dr. Heyman in Dresden began employing the ophthalmoscope regularly and agreed with Coote that it was of diagnostic value and its' use should be employed by all professing to be specially trained in eye diseases(41).

About the middle of the nineteenth century a great deal of ingenuity was expended on various alternative methods of viewing the fundus. Czermak(1851) introduced the orthoscope, or hydroophthalmoscope, wherin a glass box filled with water is fixed in front of the eye so that the corneal refraction is eliminated, thus rendering the fundus more easily visible: this led to several modifications and may be considered the forerunner of the contact glass. Coccius(1859), and afterwards Zehender(1863), showed experimentally that it is possible to see the fundus of one's own

eye, an observation which was followed by the ingenious autoophthalmoscope of Heymann(1863) by which the left eye examines an illuminated area of the right. About the same time Girand-Tenlon(1861) introduced the first practicable binocular ophthalmoscope, thus laying the foundations of stereoscopic ophthalmoscopy: while deWecker and Roger(1870) by partial deflection of the beam of light by a prism, introduced the demonstration ophthalmoscope, by which the fundus could be seen by more than one observer simultaneously.

Following this preliminary outburst of activity a period of relative stagnation lasted until the end of the nineteenth century. The renewal of interest arose from the availability of better illumination than was afforded by the petroleum or gas sources which were at first available. At an early date Macdonald(1850) had suggested the use of sunlight, and the brilliant light which may be obtained in this way is sometimes useful when the ocular media are cloudy, but the next real impetus to the development of ophthalmoscopy was due to the perfection of small electric bulbs or flash-lights; the natural sequence of this was the development of the electric ophthalmoscope, the first of which was introduced into Amer-

ica by Dennett(1885) of New York, and into England by Juler(1886) of London. The electric ophthalmoscope, however, did not come into general use until the beginning of the twentieth century(21). Since this time there has been many improvements of the electric ophthalmoscope; the highly accurate and precise instrument which we use today, being the end product of many modifications and improvements. The latest development has been made by H. Friedenwald who has during the past few years devised the instrument bearing his name. It is a high-powered instrument having a slit beam.

ANATOMY

Gray describes the retina as "a delicate nervous membrane, upon which the images of external objects are received. Its' outer surface is in contact with the choroid, its' inner with the hyaloid membrane of the vitreous body. Behind it is continuous with the optic nerve; it gradually diminishes in thickness from behind forward, and extends nearly as far as the ciliary body, where it appears to end in a jagged margin, the ora serrata. Here the nervous tissue of the retina end, but a thin prolongation of the membrane extends forward over the back of the ciliary processes and iris, forming the pars ciliaris retinae and pars iridica retinae already referred to. This forward prolongation consists of the pigmentary layer of the retina together with a stratum of columnar epithelium. The retina is soft, semi-transparent, and of a purple tint in the fresh state, owing to the presence of a coloring material named rhodopsin or visual purple; but it soon becomes clouded, opaque, and bleached when exposed to sunlight. Exactly in the center of the posterior part of the retina, corresponding to the axis of the eye, and at

a point in which the sense of vision is almost perfect, is an oval yellowish area, the macula lutea; in the macula is a central depression, the fovea centralis. At the fovea centralis the retina is exceedingly thin, and the dark color of the choroid is distinctly seen through it. About three cm. to the nasal side of the macula luteae is the entrance of the optic nerve(optic disk,), the circumferance of which is slightly raised to form an eminence(colliculus nerve optici); the arteria centralis retinae pierces the center of the disk. This is the only part of the surface of the retina which is insensitive to light, and is termed the blind spot."

"The retina consists of an outer pigmented layer and an inner nervous stratum or retina proper."

"The pigmented layer consists of a single stratum of cells. When viewed from the outer surface these cells are smooth and hexagonal in shape; when seen in section each cell consists of an outer non-pigmented part containing a large oval nucleus and an inner pigmented portion which extends as a series of straight threadlike processes between the rods, this being especially the case when the eye is exposed to light. In the eyes of albinos the cells of this layer are destitute of pigment."

"The nervous structures of the retina proper are supported by a series of non-nervous or sustentacular fibers, and, when examined microscopically by means of sections made perpendicularly to the surface of the retina, are found to consist of seven layers, named from within outward as follows:

1. Stratum opticum
2. Ganglionis layer
3. Luner plexiform layer
4. Luner nuclear layer, or layer of inner granules
5. Outer plexiform layer
6. Outer nuclear layer, or layer of outer granules
7. Layer of rods and cover."

For purposes of this thesis it is not necessary to describe the detailed anatomy of these layers.

"The nervous layers of the retina are connected together by a supporting frame-work, formed by the sustentacular fibers of Muller; these fibers pass through all the nervous layers, except that of the rods and cones. Each begins on the inner surface of the retina by an expanded, often forked base, which sometimes contains a spheroidal body staining deeply with hematoxylin, the edges of the bases of adjoining fibers being united to form the membrana limitans interna.

As the fibers pass through the nerve fiber and ganglionic layers they give off a few lateral branches; in the inner nuclear layer they give off numerous lateral processes for the support of the bipolar cells while in the outer nuclear layer they form the membrana limitans externa at the base of the rods and cones. At the level of the inner nuclear layer each sustentacular fiber contains a clear oval nucleus"(32).

The central retinal artery arises from the ophthalmic and enters the optic nerve in company with the vein, about 10-12 mm. behind the eyeball. It enters the bulb of the eye through a portion of the optic disk known as the porus opticus. The artery immediatly bifurcates into an ascending and a descending branch. The ascending branch divides into the superior temporal and superior nasal branches. The descending branch divides into the inferior temporal and inferior nasal arteries.

The temporal branches are slightly larger than the nasal, they sweep in broad curves around the macula to which they send their principle branches. The macula is divoid of visible vessels but is very rich in capillaries except at the fovea centralis which is absolutely avascular.

The cilioretinal vessels spring from the temporal side of the disk, and are distributed to the region between the disk and macula.

Only the inner layer of the retina is supplied by the retinal vessels. The outer layer(rods and cones) is supplied by the choroidal vessels.

The central vein of the retina usually empties into the superior ophthalmic. The ophthalmic vein discharges into the cavernous sinus but also communicates with the facial vein. This is important because it provides outlet for retinal blood should the cavernous sinus become clogged. In the head of the optic nerve the central retinal artery anasthamoses with the vessels from the choroid as follows: Just before the short ciliary arteries enter the choroid they give off small branches which unite and form a ring in the sclera around the optic nerve which is known as the Circle of Zinn. Branches from this enter the head of the nerve and anasthamose with twigs from the central artery of the retina. These anasthamoses are little more than capillaries; nevertheless they provide a feeble collateral circulation for the retina in closure of the central retinal artery. Vessels from the central artery supply the nerve and furnish

a rich capillary plexus which gives color to the optic disk. After emerging from the optic disk, the retinal vessels form a strict terminal system, i.e. they do not anasthamose with other vessels or with each other. The retinal vessels are provided with perivascular sheaths which are the lymph channels of the retina(52).

The central artery of the retina, according to Hertel, has a lumen of about 210 microns, narrowing to 170 microns at the lamina cribosa. In infancy the internal elastic layer lies close to the endothelium. As the arterial branches become narrower, this layer becomes thinner and can be followed as a layer of longitudinal fibers to arteries of a diameter of 10 microns. According to Friedenwald, the internal elastic lamella disappears entirely beyond the primary branches of the central artery. The media consists mainly of circularly arranged fibers, with here and there some which are arranged obliquely. It also contains some elastic fibers. The adventitia is sharply demarcated from the media and contains elastic fibers in addition to the connective tissue; it merges indistinguishably into the connective tissue surrounding the vessel. As the branches get smaller, the muscle fibers are reduced in number until they can hardly be demonstrated in histological preparations(23).

PHYSIOLOGY

Pulsation of a vein upon the optic disk is a physiologic phenomenon and is present in approximately 75 per cent of normal eyes. Donders explains it as follows "The cardiac systole drives the blood wave into the eye, producing momentary increase in intraocular pressure, and this rythmic increase in pressure may be sufficient to empty the veins on the disk. Possibly venopulsation may be assisted by pulsatory pressure variations in the cavernous sinus". In accordance with the above theories, the physiologic venous pulse is diastolic, that is, the veins fill during cardiac diastole and empty as the pulse wave reaches the eye. Another explanation of the venous pulse is that either in the nerve or in the disk, the the artery presses against the vein and the arterial pulsation is transmitted by contact. This would explain why pulsation is present in some cases and absent in others, and why it frequently is limited to a single vein. Certainly pulsation always occurs in a vein which is visibly in contact with an artery. Venous pulsation can be produced in normal eyes by lightly compressing the eye, if the pressure is in-

creased, pulsation appears in the artery and the vein collapses. Pulsation of the retinal veins may be of pathologic import. A positive or systolic venous pulse may occur in tricuspid regurgitation. In aortic regurgitation both veins and arteries pulsate. According to Oatman(1) in 1913 pulsation in the retinal arteries is always pathologic.

According to his theory, the systemic blood pressure is just enough higher than intraocular pressure to permit a smooth flow of blood through the retina without visible pulsation of the arteries. If, however, either the intraocular pressure rises above or systemic blood pressure falls below its' relative standards, the inflow of blood is hindered and the artery will pulsate. Two forms of arterial pulsation will be recognized, (1) the pressure pulse which appears only in the arteries on the disk. It occurs in the heightened eye pressure of glaucoma and in the lowered blood pressure of syncope, ischemia, etc. (2) The locomotion pulse, which is manifested throughout the retina by a distinct motion of the arteries seen wherever they make a quick bend. This type of pulse occurs in aortic regurgitation, when it is accompanied by a pulsation of the veins and by an alternate flush-

ing and paling of the disk. It may occur with aneurisms, in Basedow's Disease, and in the earliest stage of diffuse arteriosclerosis when the arteries are relaxed. Pulsatory phenomena may be stimulated by slight motions on the part of the observer or subject, and the diagnosis of abnormal pulsation should never be made unless it is distinct and unmistakable.

Friedenwald(30) in 1934 claims that pulsation in retinal arteries is not pathologic. He states that they may be seen in the majority of children. In the adult, however, the magnitude of the pulse decreases considerably and is scarcely seen in routine ophthalmic studies. Any easily visible pulsation should be regarded as possibly being pathologic, possibly representing an increase in intraocular pressure, or a decrease in systolic blood pressure with increased pulse pressure.

Keyes and Hatcher(43) in 1940 state, "A visible pulse in the retinal arteries is a common physiologic phenomenon. This varies with general and local disturbances of circulation and of the eye. Statements to the effect that pulsation of the retinal arteries is always pathologic should be corrected in textbooks of ophthalmology".

PATHOLOGY

The pathology of the retina in the various hypertensive states is so intimately related to these conditions that it is impossible to describe them without simultaneously describing the retinal findings. In view of this fact the discussion of retinal pathology per se will be rather brief and general.

The retina constitutes an organ within an organ, namely, the eye. Its' circulation is considerably independent of the general circulation and is regulated according to the needs of the retina by the vegetative system, Independent of how the circulation is maintained in other parts of the body. Disturbances in the circulation of the retina are caused here as elsewhere by vasomotor changes, by mechanical means, or by both. There is an advantage in studying circulatory disturbances in the retina over studying them in other organs in that changes in the fundus of the eye can be directly seen with a magnification of sixteen times. Although, the retina itself is transparent, the vascular tree itself, as well as any deformation of the vessels can be seen in detail; and an opaque substance, such as a transudate, a hemorrhage or a hyaline or lip-

oid deposit, interferes with the transparency of the the retina and is readily visible. The following are the circulatory disturbances in the retina which must be looked for: (1) changes in the walls of the vessels, (2) transudation (edema), (3) hemorrhages, (4) fat and lipoid deposits, (5) hyaline deposits, and (6) proliferated glial and connective tissue.

The diseases which produce the vasomotor ar mechanical changes responsible for the prestasis, peristasis, stasis, obstruction anemia, infarction and their consequences, are manifold and include, (1) pathologic changes in the walls of the vessels, (2) functional constriction of the walls of the vessels, either localized or as a part of a generalized arterial constriction, and (3) functional constriction superimposed on anatomic changes in the walls of the vessels or preceding these changes.

A few words are necessary regarding the phagocytic elements which engulf particles of hyaline, lipoid and red blood corpuscles. Their origin is not completely determined. Many phagocytes are undoubtedly histiocytes from the adventitia of the vessels. Some are perhaps glial cells. Other phag cytes have definitely been shown to be derived from th pigment epithelium

layer. The cells of this layer are probably not phagocytic, but in the presence of adventitious substances in the retina they are shed and a proliferation of new cells contain less and less pigment; they have ameboid movements and are phagocytic. The still pigmented cells gradually lose their pigment which then becomes an object of phagocytosis and is carried by the phagocytes to the perivascular spaces.

ARTERIAL HYPERTENSION

General Information

The blood pressure in a state of health is the resultant of a number of forces, among the chief of which are the contractions of the heart and the peripheral resistance provided by the arterioles, although the elastic recoil of the large arteries and the state of the capillary bed are also of importance. In spite of the fact that these varied factors are continually altering, so perfect is the coordination between the several parts of the circulatory system that under ordinary conditions any sudden change in pressure is rapidly restored to normal. Should the pressure be continuously below or above normal the condition is called respectively hypo- and hypertension.

Continued hypertension occurs as a result of a number of different conditions. Of these the two most important are acute glomerulonephritis together with the closely related toxemias of pregnancy, and the form known as essential hypertension, so-called because it is not dependent on disease of the kidneys or of any known organ. The latter form is by far the more common. Hypertension associated with arteriosclerosis also forms a group which is considered as

a separate entity by some men. It should be remembered that arteriosclerosis is generally found to a greater or lesser extent in all individuals in the old-age group and so may be associated with the above-mentioned groups, namely: acute glomerulonephritis and essential hypertension. The hypertensive states to be discussed in this thesis, with special reference to retinal vascular changes are essential hypertension, arteriosclerosis, and the toxic hypertensive syndrome of pregnancy.

ESSENTIAL HYPERTENSION

Pathogenesis

In following the course of the blood pressure in a person in whom essential hypertension eventually develops the following sequence of events is observed: In middle life, that is, in the forties or earlier, and in an occasional young person, there are noticed a loss of stability of the normal arterial pressure. There are periods of variation in which the blood pressure rises to 160 mm. and higher, only to return to normal at other periods. The rise in pressure may last for days. Normal persons of that age will have fluctuations of blood pressure from day to day depending upon exercise, diet, nervous tension etc. and

really the variations in blood pressure seen in essential hypertensives are but exaggerations of these normal fluctuations. With the passing of time, varying from months to years it will be noticed that the pressure readings become progressively higher and last over longer periods of time. Usually these readings go to about 160 or 170 mm. of mercury. With the passing of a further period, the greater variability is still present or is even more marked, but now the blood pressure is permanently high, perhaps around 200 mm. or higher. The increase in wear and tear as a result of the persistently high blood pressure has exercised its' influence on th arteries, and both the aging and the arteriosclerotic processes have become intensified, and pathologic changes in the arteries are noticable. It is evident that the mechanism for maintaining normal blood pressure is defective in this type of patient. There is no known external factor or any general disease which causes the defectiveness. The only factor known is that of heredity.

Elwyn(23) states as follows: "I define essential arterial hypertension as a disease in which there occurs in some persons in middle life and occasionally earlier a loss of stability in the mechanism for the

maintenance of normal arterial pressure. This loss of stability is the result of an inherited defect already present in the germ plasm. It manifests itself at first by variations in the height of blood pressure which are exaggerations of the normal variations. Eventually there is a complete loss of ability to maintain the normal blood pressure, which remains now at a high level. With the persistence of the high level of blood pressure, the aging and the arteriosclerotic processes are hastened and appear earlier in life".

Vascular Changes

Since essential hypertension is a disease which begins with a variability in the height of the blood pressure in middle life and extends into old age, it is obvious that any secondary arterial changes will vary with the duration and the degree of the hypertension. In the early years there are practically no changes in the arterial system beyond the ones appropriate for the age period. As the years pass, the aging and the arteriosclerotic processes become more and more apparent. These degenerative processes particularly affect the vessels of the kidneys. These vessels may be exclusively affected in some persons;

in others they are affected long before the rest of the arterial system. For this reason essential hypertension is also spoken of as benign renal sclerosis.

Pathological changes in the vessels of the kidneys which characteristically accompany this disease are as follows according to Elwyn(23):

1. "In the medium-sized and small arteries there Is a thickening of the intima consisting of a multiplication of the elastic lamella with an increase of connective tissue between the lamellae. The muscular layer is reduced in thickness. This process is diffuse throughout the vessels. In the hypertrophied intima there occur regressive changes, namely, hyaline and lipoid deposits in an irregular focal distribution.

2. In the arterioles there are deposits of hyaline and lipoid which cause a thickening of the walls of the vessels. These deposits are not diffuse throughout all the vessels but are formed in some and not in others, and in those affected they are not present throughout the whole vessel. These deposits increase in extent with the duration of the hypertension."

Other arteries in the body may be free from any disease. However, with the progress of hypertension the arteriosclerotic process begins to appear here

and there in the other arteries. In many instances the arteriosclerosis is widespread, affecting the aorta and its' branches, the arteries of the heart and of the brain. The arteriosclerotic process is found most frequently in the kidneys only; in some cases and in those in the advanced stage it is found also in the pancreas, less frequently in the liver and occasionally in other organs. The arterioles of the extremities are not affected.

Retinal Changes

In the early stages of essential hypertension, and sometimes even in the advanced stages, there are no changes in the vessels of the retina to indicate a premature aging or the presence of arteriosclerosis. In many cases, perhaps in most, the prolonged increase in wear and tear affects the vessels of the cerebral circulation and with it the arteries of the retina. There is then noticed ophthalmoscopically the premature appearance of certain signs in the retinal vessels. These according to Behan(9) are:

1. "The vessels have the appearance of uniform distention and fullness.
2. "The light streak is broadened out; it may be greatly increased reaching almost the whole breadth of the vessel.

3. The light streak is very much brighter than normal, the brilliancy increasing with the increase of tension, until with very high tension it becomes like bright copper wire (not silver wire).

4. The tight arteries indent the veins; with medium-high tension they indent them slightly, with very high tension they indent them deeply, leading to back-pressure and all its consequences.

According to Elwyn(23) there is nothing in these signs which is absolutely characteristic of essential hypertension. It is rather their early appearance and their manifestation to an unusual degree which calls attention to the possible presence of arterial hypertension.

Roesler, Gibson and Hussey(61) studied eighty carefully selected cases of essential hypertension from the point of view of retinal vascular changes and electrocardiographic alterations and 59 per cent of them as to radiologic heart size, and correlation between these criteria was carried out.

Retinal vascular changes were noted in all of the selected cases, with 88 per cent graded as sclerosis, hypertensive type. The electrocardiogram revealed

final deflection changes in 68.8 per cent. A slight to moderate enlargement of the heart was noted in 50.8 per cent, and a marked degree in 27.1 per cent.

They concluded that there is a trend toward a positive correlation between electrocardiographic alterations and the degree of cardiac enlargement. This correlation was not demonstrated to be of high statistical significance but they offered as possible causes for this, irregular distribution of the vascular processes and the lack of strict paralellism between the systemic and central retinal artery blood pressure. They feel that inasmuch as there is inadequate correlation between the three criteria in question, it seems desirable to have, in a given case of essential hypertension, an evaluation of the eyegrounds, electrocardiogram, and the heart size, in addition to more routine studies when one attempts the difficult task of making a practical prognosis for a patient who has this disease.

Shelburne, Hawley and McGee(65) in a recent article have shown the relationship between arteriovenous nicking and enlargement of the heart in ambulatory patients with hypertension. They chose this particular retinal finding because (1) arteriovenous nicking is

easily and accurately identified after one has had sufficient practice, (2) it is the most constant change present in all forms of long-standing hypertension and (3) it never occurs except in patients who have or have had hypertension. They were able to show by a study of 317 patients with hypertension that arteriovenous nicking is closely related to enlargement of the heart; that if this lesion is found the patient may also be expected to have cardiac enlargement. They also concluded that if arteriovenous nicking and enlargement of the heart are found simultaneously, the latter may be accounted for by hypertension even if the blood pressure is normal at the time of examination. They also feel that if the heart of a hypertensive patient is enlarged, And no arteriovenous nicking is found, the enlargement is not likely to be due to hypertension alone and a careful search for other lesions, such as those of severe coronary arterial disease, syphilis or rheumatic fever, is clearly indicated.

The following table is copied directly from their article and clearly shows the basis for their conclusions:

Table I-- Incidence of arteriovenous nicking with

and without associated heart enlargement.

	No. patients	%
Nicking present, Heart enlarged.....	142	95.7
Nicking present, heart normal size.....	6	4.3

Yater(76) studied 137 cases of heart disease and reached the following conclusions: "In 96 per cent of cases due to hypertension alone there were characteristic changes in the retinal arterioles. Therefore in hypertensive heart disease the retinal examination is important. It is particularly important in those cases in which the blood pressure has returned to normal. In this type of case the retinal findings may constitute the final diagnostic criterion.

In 92 per cent of cases of heart disease due mainly to coronary arteriosclerosis there were retinal vascular changes either of the hypertensive type or of the senile fibrosis type.

It should be remembered that about one-half of all the cases of hypertensive heart disease develops coronary arteriosclerosis. Retinal arteriosclerosis in the presence of heart disease means that the heart disease is due either to hypertension, coronary arteriosclerosis, or both, provided there is no specific evidence of other causes of the disease of the heart. If the

retinal changes are of the type associated with decrescent arteriosclerosis and there is no clinical evidence of other cause of the heart disease, it is probably due to coronary arteriosclerosis, without antecedent hypertension. If neither type of vascular retinal change is present, the chances are against the heart disease being due to either hypertension or coronary arteriosclerosis. If prognosis is rendered more grave by the presence of retinitis or neuroretinitis an early death can be anticipated".

In malignant hypertension the ophthalmoscopic findings according to Elwyn(23)are: (1)"Signs of advanced arteriosclerosis and arteriolosclerosis, consisting of angular tortuosity of the vessels, especially the small ones, variations in caliber of the lumen, apparent constriction of veins where they are crossed by arteries, and a sharp and bright light reflex with irregularity in its' width; (2) arterioconstriction, as shown by the narrowed arteries, especially the smaller branches; (3) signs of local circulatory disturbances of acute character, consisting of edema, hemorrhages, cotton wool patches, blurring of margins of the optic disk and distinct papilledema, and(4) sharp white spots of hyaline deposits,

glistening spots and the star figure in the macular area.

The whole clinical picture in the fundus represents the local circulatory disturbances as a result of a persistent and varying arterial constriction which has suddenly been added as a malignant factor in the course of essential hypertension".

ARTERIOSCLEROSIS AND HYPERTENSION

Retinal Arteriosclerosis

Histologic studies of the central artery have been made by the German writers Raehlmann, Hertel, Harms, and Baumgartner. Bridgett(11) and Friedenwald (29) in America have also made valuable contributions. They are fairly well agreed that the development of the artery throughout life corresponds to that of other arteries of the same size. There is a normal physiologic thickening of the intima in the ascending period of life consisting of elastic fibers and interstitial tissue, probably containing muscle fibers. This remains without much change during the stationary period of life, that is, until about the end of the fourth decade. The aging process begins in the central artery as in other arteries, after forty-five years of age. This process consists of a thickening of the internal elastic lamella into two or three layers and in an increase of connective tissue. Bridgett in a study of the central artery of the retina at 200 autopsies found that during the third and fourth decades it was usually but not always possible to find a thin, almost homogeneous subendothelial band as well as separation of the internal elastic lamella into a

double, and more rarely, into a triple layer. He also found a slight increase in the elastic tissue in the adventitia. At 45 autopsies he found the development of the subendothelial tissue somewhat excessive.

The aging process seems thus to develop in the central artery of the retina in the same manner as it does in other arteries; the arteriosclerotic process is added to this aging fibrosis. The arteriosclerotic process begins here also with the appearance of fat and lipoids in the intima. This is followed by the formation of atheromas, rupture of the elastic fibers and proliferation of subendothelial connective tissue; calcium is deposited in the atheromatous areas. Nodular thickening of the walls of the vessels and deformation of the vessels are the results.

The changes in the ophthalmoscopically visible branches of the central artery are similar to those of the main artery but are less common. Friedenwald(29) pointed out "that the ophthalmoscopically visible lesions are merely fringes of a lesion, the major portion of which is obscured from view." In the smallest arteries and arterioles, which have no elastic fibers and are not visible ophthalmoscopically the arteriosclerotic process is not found. Here there are found

hyalinization and lipoid deposits such as occur in the arterioles of the other organs such as spleen, kidneys, pancreas and brain.

Ophthalmoscopic Appearance of Retinal Arteriosclerosis

In examining the retinal vessels one must keep in mind that the aging process is the same as in other vessels of similar size. It manifests itself in dilitation and tortuosity of the vessels, to which is added the irregularly distributed arteriosclerotic process. One must also keep in mind that when looking at a normal vessel with the ophthalmoscope, only the blood column is seen and not the wall of the vessel. When there are irregular and patchy changes in the wall they produce an irregularity in the appearance of the blood column. There are, therefore, as a result of the aging plus the arteriosclerotic process, the following signs:

1. A tortuosity of the wall of the vessel. This tortuosity differs from that seen in hypermetropic eyes of young persons and from the congenital variety in that it is more or less angular and does not have the sinous quality found in the other conditions. In cases of advanced retinal arteriosclerosis it causes the small vessels in the macula to have a corkscrew

appearance.

2. Localized variations in the calibre of the vessels. This change is the result of localized arteriosclerotic patches. There are all kinds of constrictions in caliber, single or multiple, extending over variable distances, with normal appearing stretches in between the constrictions or with dilatation between them.

3. Localized arteriovenous constriction. This sign was first observed by Gunn(34). It is seen as an indentation or compression of the vein where it is crossed by an artery. The arteriovenous crossing has been studied histologically in a number of patients of all ages with or without hypertension. It has been found that the artery and vein lie close together and have a common adventitial coat at this point, an observation made by Friedenwald(25). The vein dips down deep into the retina where it is crossed by an artery, in young persons moreso than in older ones. In old persons with or without hypertension there is a thickening of the wall ao the vessel and of the common adventitial coat. As the vein dips down, it is hidden by the thickened or sclerosed non-transparent wall. Gunn's sign is thus a sign of thickened or sclerosed

vessels.

4.Widening of the arterial light reflex. H. Friedenwald(25) pointed out that the width of the light reflex is closely related to the width of the blood column. It would, therefore, be wider in the dilated arteriosclerotic vessel. Behan(9) states that diminution in the size of the vessel and a 'silver wire reflex' shows advanced sclerosis, and offers this as a characteristic which differentiates these arteries from those of essential hypertension in which the reflex is the 'copper wire' type.

These signs are found in all degrees of variation. In evaluating them it must be kept in mind that while arteriosclerosis occasionally occurs in young persons, below forty years of age, it is commonly seen as an addition to the aging process. As the latter begins to manifest itself toward the end of the fifth decade, and as both it and the arteriosclerotic process are slow in developing, it is obvious that the signs of arteriosclerosis will become noticable only in advanced age, in the sixth decade and beyond. This is the normal occurrence. When these signs are found they give visible expression to the degree of the arteriosclerotic process in the retinal vessels them-

selves; since the central artery of the retina is a part of the cerebral circulation, the signs are a valuable guide to the approximate degree of arteriosclerosis in the brain, and the degree of arteriosclerosis in the retina gives some indication as to the degree of arteriosclerosis in the body generally. This is especially true when these signs occur in comparatively young persons, that is, in the fifties or forties, or even in younger persons. The wear and tear which blood vessels can withstand are principally dependent upon their inherited characteristics, and the signs of wear and tear usually become manifest in the old-age group. When signs of a considerable degree of arteriosclerosis of the retinal vessels are encountered before the senium and in young persons, two etiologic factors must be considered as responsible: first, an inherited deficiency in the wall of the vessel, causing it to give way sooner to the ordinary wear and tear; and, second, an increase in wear and tear, causing the vessel to give way prematurely. The one important factor which increases the intensity of the wear and tear of the arterial wall is a persistent increase in blood pressure, especially essential hypertension. The finding of signs of arterio-

sclerosis in the retinal vessels of middle-aged and of young persons calls, therefore, for an immediate investigation as to the presence of persistent arterial hypertension. Here according to Elwyn(25) it is important to make clear distinctions. He states "These commonly accepted signs, angular tortuosity of vessels, variations in caliber, apparent arteriovenous constriction and widening of the arterial light reflex, are not signs of general arterial hypertension. They are signs of arteriosclerosis of the retinal vessels. Only their appearance in middle-aged or in young persons serves notice to the observer that something is responsible for their premature occurence, and the presence of a persistent hypertension may be surmised and must be sought for."

It should be bourne in mind that the signs of arteriosclerosis are permanent. There is no regression of the sclerotic process whereas signs of essential hypertension vary with the blood bressure.

According to Davis(19) the arteriosclerotic process may be discovered earlier by ophthalmoscopic signs than in any other manner. He claims that many people in middle life who come to the ophthalmologist complaining of eye weakness or eye strain have early

signs of arteriosclerosis in their retinal vasculature. This information would be of unlimited value to internists, neurologists, and neruological surgeons. Davis feels that in all liklihood the toxins causing arterial hypertension probably attact by predilection the carotid system as the luetic toxins do the aorta. This is why the hyperpietic patient is more prone to hemorrhage in his head than in other parts of his body.

TOXIC HYPERTENSIVE SYNDROME OF PREGNANCY

Mussey(50) in 1936, examined the retinas of 108 patients who had acute toxic vascular(hypertensive) syndrome of pregnancy. In 72 percent of cases in which the systolic pressure was 140 mm. of mercury or more, positive evidence was found of more or less change in the retinal arterioles or in the retina proper. As a rule, changes in the retina, which at first consisted of spastic norrowing of the retinal arterioles, revealed a definite increase in degree and severity, with increase in the height of systolic blood pressure and the duration of the toxemia. Information obtained by the retinal examination of patients who have acute hypertensive toxemia of pregnancy was considered to be a distinct aid to the obstetrician in determining if, and when, pregnancy should be terminated. It seemed evident that when the spastic condition of the retinal arterioles was maintained, the first appearance of cotton-wool exudates and hemorrhages in the retina indicated the danger of permanent systemic arteriolar injury. He concluded that in the presence of such retinal changes, pregnancy should be terminated promptly.

In 1938 Shultz and O'Brion(63) made a survey of 47 cases of hypertensive toxemia of pregnancy which occurred in 2,365 pregnant women and the following conclusions were drawn:

Ophthalmoscopic examinations of the fundi were of value in the diagnosis, prognosis, and management. Retinal changes should be added to the diagnostic triad; that is, hypertension, albuminuria, and edema. Approximately only one-third of the patients complained of visual disturbances.

Retinal changes appeared to more or less parallel the severity of hypertension and thus of the toxemia: in those with normal fundi the average blood pressure was 105/109; those with spasm of the retinal arterioles showed an average blood pressure of 170/108; in the group with angiosclerosis, the average blood pressure was 181/118; and those with retinitis had an average blood pressure of 199/137. Patients with normal fundi or angiospasm rarely showed permanent damage of the vascular system or kidneys at the examination four months after delivery. Those with angiosclerosis or retinitis frequently showed signs of permanent damage at that time. Hence, the prognosis is not good, as a rule, for those with organic changes in the retinal

vessels or retinitis.

The retinal changes did not parallel either the albuminuria or edema. Patients with retinitis almost invariably showed an increase in the number and extent of retinal lesions for a few days following delivery. They deduced that patients with normal fundi or agniospasms may be treated conservatively, furthermore, that the fundi should be studied frequently and, if indications of organic change in the vessels or retina appear, the uterus should be emptied. In cases of angiosclerosis of the retinal vessels or retinitis it is safer to empty the uterus at once.

As these two reports might indicate, the majority of writers in recent years, are agreed that pathologic and clinical findings in cases of acute late toxemia of pregnancy show evidence of more or less generalized vascular disease, primarily a disease of arteriolar spasm throughout the body. Acosta-Sison(2) and others have presented evidence that vascular damage is the basis of the pathologic changes in the liver, kidneys, brain, and heart. Likewise, examination of patients, suffering from severe non-convulsive toxemia(pre-eclampsia) and convulsive toxemia(eclampsia) usually demonstrates the presence of spasm of the small arter-

ies of the retina and of the capillaries of the nail folds. Futhermore in most instances, accurate retinal examination will reveal a distinct difference between the retinal changes produced by chronic vascular sclerosis or chronic nephritis and those present in cases of acute late toxemia of pregnancy. Cheney(14), Wagener(71), Hallum(37), and others reported that changes often occurred in previously normal vessels in the course of late toxemia of pregnancy. Wagener made the important observation that acute vascular changes in the retina were sometimes superimposed on lesions characteristic of chronic vascular disease.

In a broad sense, the degree of severity of acute hypertensive toxemia is measured more by the height of blood pressure than by any other symptom or finding. Also, permanent vascular and glomerular change which may follow acute toxemia seem to depend on the height of the blood pressure and the length of time during which the hypertension, or the toxin producing it, is imposed on the vascular system. Follow-up studies, notably by Peckham, Herrick(39), and others have shown that chronic cardiovascular disease or chronic glomerular sclerosis occurs among many women as a direct consequence of acute hypertensive toxemia. According

to Peckham(54) and Stout, repeated examinations of patients whose condition was diagnosed as low reserve kidney(considered by many to be mild late toxemia of pregnancy) revealed that five years later, approximately one-half of these patients had evidence of chronic arterial disease or chronic nephritis. This incidence rises with the height and duration of the hypertension, the condition being notably frequent among those whose systolic pressure registered more than 200 mm. of mercury.

Discussion of the management of acute hypertensive toxemia must include, also, consideration of chronic cardiovascular disease and chronic nephritis which ante-date pregnancy. When definite chronic nephritis or extreme hypertension is susceptible of diagnosis early in pregnancy, management of such a case ofter differs sharply from that of acute hypertensive toxemia, the symptoms of which appear later in pregnancy. Retinal examinations deserve special mention here, because evidence of chronic retinal changes or nephritic retinitis may be demonstrable even when the results of tests of renal function are not diagnostic. If pregnancy is complicated by pre-existing chronic vascular sclerosis or chronic nephritis, there is a marked tend-

ency toward exacerbation of the pre-existing disease in the first, or early in the second trimester, and continuation of pregnancy usually results in a much more serious condition than before. Sometimes patients who have an occult nephritis, which may not be suseptible of definite diagnosis prior to pregnancy and some who have mild hypertension may pass through pregnancy without suerimposed toxemia.

Dr's. Mussey and Mundell(51) reviewed the pathogenesis of conditions classified as late toxemia of pregnancy, because the concept that more or less prolonged, acute, general, vascular spasm is responsible for permanent cardio-vascular and glomerular injury, which may follow toxemia of pregnancy, has emphasized the need for the termination of pregnancy in certain of these conditions. In addition to readings of the blood pressure, results of examinations of urine, evaluation of subjective symptoms and, occasionally, chemical examinations of the blood, a valuable index to the degree of spasm of the vascular system and the rate at which this spasm is progressing, is furnished by changes observed in the vessels of the retina. Masters found that, in every case in which the blood pressure was elevated to 150 mm. of mercury or more, there was narrowing of the caliber of the retinal

arteries. Among 44 cases of toxemia, he observed this retinal change in three cases in which the blood pressure was initially less than 150 mm.of mercury but subsequently rose much higher. Wagener(73) found there were retinal changes in 70 percent of pregnant women whom he examined, and whose systolic blood pressure was more than 140 mm. of mercury. Later Hallum(56) quoted similar findings.

Wagener(73) also states: "In cases of toxemia of pregnancy in which there is an associated rise in blood pressure, changes in the arterioles appear first and those in the retina proper, commonly called "retinitis" are secondary to and apparently dependent upon the changes in the arterioles. The caliber of the arterioles appears narrowed and the lumen is reduced because of spastic contraction and increased tonus of the walls of the arterioles. This change in the arterioles may disappear entirely if there is early and permament fall in blood pressure. The constriction soon becomes fixed if the toxemia progresses. When the constriction of any arteriole becomes so fixed and severe as to cause secondary capillary ischemia or stasis, localized edema and hemorrhage appear in the adjacent retina. If the

toxemia continues, this spastic constriction may become so generalized and severe as to produce diffuse retinitis of the albuminuric type, the classic "retinitis of pregnancy nephritis". The presence or absence, or the advancement of the involvement of the arterioles can be determined best by frequent systemic examinations of the retina.

It has been found that Wagener's description of the retinal changes in cases of acute toxemia of pregnancy could be divided into four stages or grades, related to the severity and duration of the hypertension: Grade I, the first sign of which is spastic narrowing of the arterioles of the retina, which may affect all branches of the central artery: Grade II, in which irregular lumina of the arterioles usually appears first or to a more severe degree in the smaller nasal branches, sometimes varying in situation or degree from day to day: Grade III, in which narrowing and constriction are more fixed and individual cotton-wool patches and hemorrhagic regions may appear in the retina: and Grade IV, in which diffuse retinitis of the albuminuric type is found. By the use of this system of grading of progressively severe retinal changes, a tabulation previously reported, was made

of the retinal examinations of 108 patients who had late toxemia of pregnancy; in 14 of these cases, there was pre-existing hypertension. The data indicated that (1) all patients who had a systolic pressure of 200 mm. of mercury or higher had evidence of acute retinal changes, most of them severe, (2) 90 percent of the patients who had a systolic blood pressure between 170 and 200 mm. of mercury had retinal changes, which were predominantly mild in character. It was shown also that there was a progressive relationship between the higher systolic blood pressure and more severe changes in the retinal arterioles. Comment was made that, in a number of cases, the retinal arteriolar changes were the deciding factors in determining if, and when pregnancy should be terminated. Hallum (36) states, "The obstitrician will find that a study of the eyegrounds, when considered with other signs of toxemia, will be a real aid, probably the most consistently reliable guide in determining when pregnancy should be terminated."

Mussey and Mundell(51) state, "For the past fifteen years, information obtained from examination of the retina has been increasingly useful to us in the management of late toxemia. This information often

has been undertaken when retinal examination gives evidence of advance in the vascular lesion, although there has been no untoward change or, perhaps, apparent improvement has occurred in the patient's clinical condition. Induction of labor on this additional evidence, we believe, has averted an increase in severity of toxemia in a number of cases. In other cases, interference has been avoided when retinal examination has revealed either a normal retina or, on repeated examination, no advance of the acute vascular spasm, or perhaps evidence often has permitted further observation and delay, thereby increasing the chances of survival of the fetus without needlessly jeopardizing the mother."

These authors recorded results of retinal examination in a series of 107 patients who had hypertension and other symptoms of late toxemia of pregnancy. Examination of 117 of these patients revealed varing degrees of arterial spasm characteristic of acute toxemia, 23 had evidence only of chronic vascular sclerotic change, and 27 gave no demonstrable evidence of a retinal lesion. Nearly all of the last group had only mild symptoms of toxemia. Including those patients with chronic changes, 83 percent on examination

gave evidence of retinal involvement. The percentage of cases showing retinal changes increased directly with the height of the blood pressure. The incidence of patients whose retinas had undergone changes, with corresponding blood pressures is as follows: 51 per cent, never more than 150 mm. of mercury, 84 percent, between 150 and 169 mm., 92 percent, between 170 and 199 mm., and 100 percent, 200 mm. or more.

Retinal examination was of diagnostic value in 120 cases or 85% of the entire series. The degree of vascular spasm was not the sole detemining factor upon which the decision as to whether or not to interupt pregnancy was based. Other symptoms frequently indicated the termination of pregnancy. However, there were many cases of late toxemia in which a drop in blood pressure and lessened edema occured following rest, sedatives, and diuresis. The blood pressure was not alarmingly high and the patients did not feel particularly ill. Retinal examinations carried out frequently, sometimes daily, in such cases may reveal progressive changes, which indicate a similar advance of generalized arteriolar involvement before the appearance of a further rise in blood pressure or other significant symptoms. In 51, or 43.5 percent, of the 117

cases, evidence of a progressive increase in retinal arteriolar spasm was an influencing and, frequently, a deciding factor in the decision to terminate pregnancy. Among 22, or 18 per cent of the cases, the retinal changes were stationary ar had regressed. These findings influenced the decision to wait before terminating pregnancy in the interest of the fetus. Also, among twelve patients who had blood pressures ranging from 150 to 200 mm. of mercury, the absence of any vascular changes influenced the decision not to terminate pregnancy. Among some of the 23 patients who had pre-existing hypertension, many of whom were seen in early pregnancy, the differential diagnosis between chronic arterial disease and acute toxemia was made possible by retinal examination which revealed chronic rather than acute vascular lesions.

Davis (19) " Retinitis should not be allowed to develop in pregnant women. Repeated ophthalmoscopic examinations should be made to follow the development and course of the spastic lesions in the retinal arterioles. If the spastic lesion becomes more marked, termination of pregnancy should be advised at the first indication of localized hemorrhage or of exudate into the retina. The beginning of retinitis marks the onset

of irreparable organic changes of the arterioles and a few days delay may result in considerable injury. The results of this injury may not become manifest for a period of years. As a result of the progressive disease produced in the kidneys and arteries, there will be a gradual failure of health. The life of the patient may be shortened many years. In many cases vision may be seriously impaired or lost, if the pregnancy is not terminated in time. A very great responsibility rests upon the consulting ophthalmologist in such cases, as to conservation of both life and vision."

Evidence from all available sources indicates that in the toxic hypertensive syndrome of pregnancy examination of the eyegrounds is of unlimited value in determining the degree of vascular involvement and helps the clinician to determine more exactly the condition of his patient. This may be a life-saving proceedure for both mother and fetus. Routine ophthalmoscopic examinations also aid in determining the type of therapy indicated.

CONCLUSION

In this presentation the history of the science of ophthalmoscopy has been reviewed, as well as the development of the ophthalmoscope itself. In the light of what has been achieved in this field to date, it is interesting to go back through the years of its' infancy and realize that the best efforts of many brilliant and far-sighted men have been responsible for its' present-day high state of development.

The principle object of this thesis has been to show the relationship between ophthalmoscopic findings in certain hypertensive states and the general condition of the patient. It is my belief that this should give considerable evidence of the importance of examining the eyegrounds in every patient showing signs of vascular disease. It is furthermore contended that every medical practitioner should be equipped to examine the fundus and should do so at every opportunity in order to perfect himself at the art. He should realize that by so-doing he will ultimately be giving his patients greater assurance of correct diagnosis. He will also be able to more accurately determine the prognosis, and institute proper therapeutic measures.

This is well confirmed in the words of Bedell(8) "The background of the eye is the stage on which many of the tragedies of life are enacted. By careful, repeated examinations, early changes can be detected and steps taken to eradicate or diminish the ravages of destructive forces".

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